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SINUS ARRHYTHMIA, ASSOCIATED WITH ANGINAL ATTACKS OF A VASO-MOTOR TYPE.

By KENNETH H. STOKES.

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We are accustomed to think of sinus arrhythmia as an irregularity belonging to childhood and early adult life. The mechanism in this example of disordered cardiac rhythm was first considered and carefully described by Mackenzie[1] under the title of "Youthful irregularity." It is of very common occurrence in children at or about the age of puberty, when the pulse finally decreases in rate and assumes the rhythm which persists during the remainder of adult life. As a rule it is definitely related to respiration. In those who manifest it, the long pauses occur with the natural expirations, the quickening with inspiration. In the adult, there is commonly no trace of alteration in the frequency of the pulse during quiet respiration, but with forced or laboured breathing, alterations of rhythm are well known to occur. In dogs, respiratory irregularity is the rule, and is well marked even when the animals are asleep. Both in the human subject and in the lower animals, the irregularity of rhythm is universally attributed to waxing and waning of vagal tone, for it is completely abolished by section of the vagi in the latter. In the healthy adult, these disturbances of pulse frequency are practically unknown in a marked form, but as Mackenzie stated, they are not uncommon subsequent to febrile attacks or in adults upon recovery from broken compensation.

The sinus irregularity which is here described is, so far as I know, almost unique in its grade ; it is associated with slow action of the heart and with frequent attacks of an anginal nature. I am indebted to Dr. Sidney Martin for opportunities to investigate the case and for permission to place it on record, and to Dr. Lewis for the curves and their interpretation.

History, etc.

H. M., aged 36, a companion and a single woman, was admitted to University College Hospital on 20th April, 1909, for observation upon the condition of her heart. She had large fibroids of the uterus associated with irregular catamenia and considerable uterine pain. The main purpose of

her stay in hospital was to ascertain the desirability of operative interference with these tumours.

The family history is of little interest. The father died of heart failure, and the mother of consumption. Her two brothers are both dead of causes unknown; there were no sisters.

Previous History.—At 10 years of age she had rheumatic fever, and when 12 years old typhoid fever. One year ago she was laid up with pleurisy. In habits she has been quite temperate; her social condition is good, her work light, but the hours of work long.

The present illness.—Her present symptoms have been of ten years' duration, and during that time she has suffered from attacks of pain in the left side of the chest, associated with severe palpitation and faintness. The pain starts in the precordial region and radiates from this situation to the left shoulder, down the inner side of the upper arm, extending down and across to the radial side, into the thumb and adjacent fingers. The pain is associated with considerable anxiety and distress, and a sensation of constriction in the chest and inability to draw the breath. At times, and in the severer attacks, the radiation is more extensive, both sides of the neck are affected and also the right side of the chest. The pains are only acute during the attack, but soreness persists and is accompanied by hyper-sensitiveness of the corresponding areas of skin for 24 to 36 hours subsequently. Numbness and tingling of the fingers of the left hand are almost continuously present. The attacks are very frequent but variable in their occurrence; thus there may be intervals of weeks or months over which none are experienced, while at other times they succeed each other within seven, four or even two days. They usually start in the evening and are of about 10 minutes' duration; they vary greatly in intensity. She does not think that they have been more frequent of late, and knows of no definite cause to which to attribute them. She has noticed that her hands become white and insensitive directly before and during the attacks. She recognises their imminence by this means and by a general sense of uneasiness and distress which may be present for some little while prior to the onset of the more severe symptoms.

Condition.—The general impression conveyed upon examination was that of a patient with a delicate constitution, and an excitable temperament. She was comfortable and in no distress. There were no signs of broken compensation. The pulse was slow (45 to the minute), and markedly irregular; the arteries were slightly sclerosed and the tension somewhat raised (S. B. P. 150 mm. Hg.*). The heart's apex beat was in the fifth interspace, 4 inches from the midsternal line. The left limit of cardiac dulness was 4 inches, the right limit 0 inches from the same point. The cardiac sounds were clear and loud, and at the apex a blowing systolic murmur was present. The lungs were free from abnormal physical signs. Abdominal and pelvic examination confirmed the diagnosis of uterine fibroids. The urine was normal and nothing was detected in the nervous system.

* Reading with Martin's modification of Riva Rocci's instrument.

Special examination of the heart.

Numerous polygraph curves were obtained and are illustrated by the accompanying figures.

The irregularity as it presented itself at first is illustrated by Fig. 3, and consists of short and long beats placed in haphasard relation to each other. Measurements of the beats show that where a long pause succeeds more than one shorter pause, the length of the longer pause is approximately or actually double that of the pause directly preceding it. This is shown in two places in the figure. Beats which succeed the longer pauses are invariably somewhat increased in length as compared to the shorter beats already referred to.

The tracing was taken during a period of suspended breathing. On most subsequent occasions when curves were taken, the irregularity showed a definite relationship to respiration, but otherwise exhibited much the same characteristics. An example is given in Fig. 1, and the tracing includes a simultaneous respiratory curve. To the left the breathing is natural, and near the summit of each inspiration a shortened pause occurs. With forced breathing, as seen in the last part of the same figure, the irregularity maintains its relationship to the breathing, but is much more marked. While the relationship to respiration was beyond question, it was not invariably a pure respiratory arrhythmia; thus Fig. 2 shows two phases of more rapid pulsation, and, on the suspension of breathing, the heart becomes regular and slow; but at one point a shortened pause disturbs the otherwise almost regular rhythm. The same relationship to respiration is depicted in Fig. 4, in which the long pauses occur at every third beat.

In Fig. 5 they are present at every fourth beat, and in these two figures, the relationship of the length of the long beat to the length of the short beat directly preceding it, or to the two short beats in the case of Fig. 5, is very striking. In both these curves, too, the beat succeeding the lengthened pause is invariably increased in length.

The mechanism, so far as it has been examined, appears to consist of a normal rhythm intercepted by longer pauses of variable frequency in their incidence. On rare occasions, long intervals occur, over which long pauses alone are met with. An example is given in Fig. 6, where the pulse rate is approximately 39 per minute. At other times with natural breathing, the pauses in the radial pulse are of very irregular length, and the longest (Fig. 7) show no relationship to the preceding pauses. At the same time, a similar general type of irregularity is present, for each of the notably long pauses is succeeded by two or more beats of a rhythm in which the rate is gradually increasing. It is apparent that we are dealing with an arrhythmia of a very definite type, but which is complicated by more than one factor. It is an irregularity in which auricle and ventricle always participate to a like extent, for each beat of the ventricle is preceded by an auricular wave, *a*,

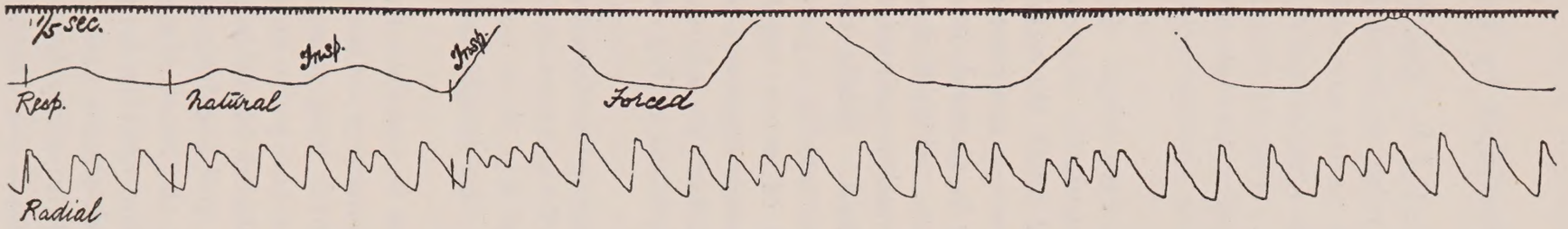

Fig. 1. × $\frac{7}{10}$ linear. Respiratory curve and radial pulse. Natural and forced breathing. The tracing shows the dependence of the arrhythmia upon respiratory reflexes. The short pauses fall with inspiration, the long with expiration and the pause.

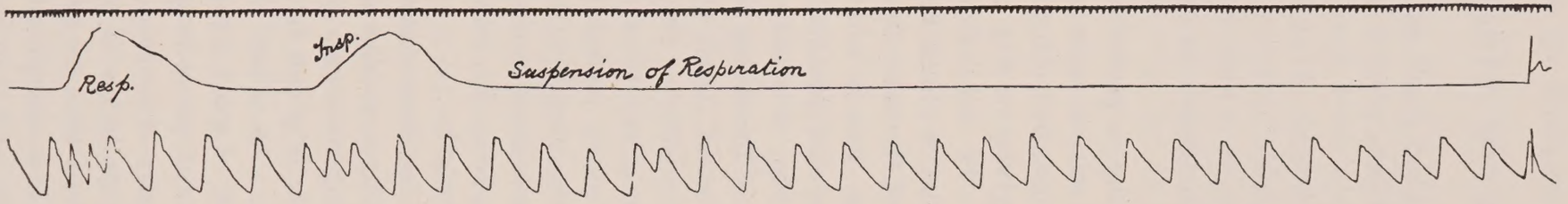

Fig. 2. × $\frac{7}{10}$ linear. Respiratory curve and radial pulse. During natural breathing and during a suspension of respiration; a single short pause interrupts the last period.

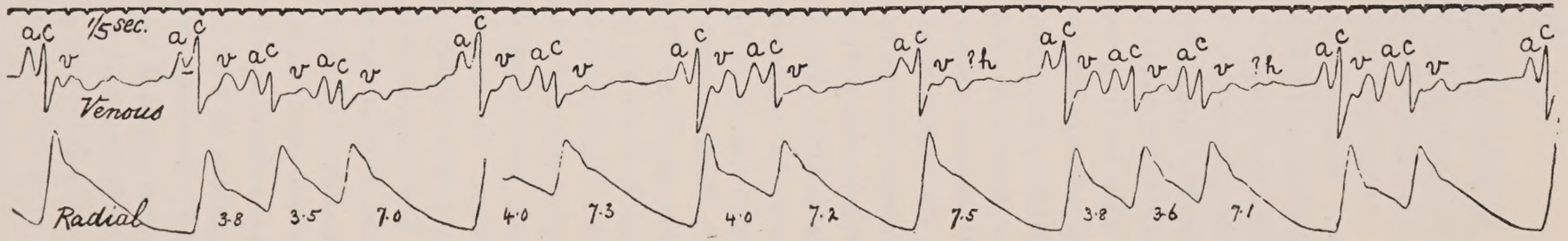

Fig. 3. × $\frac{7}{10}$ linear. Venous and radial curves showing the analysis of the heart's mechanism. Each beat is accompanied by *a*, *c*, and *v* waves. Breath held. The long pauses occur after one, two, or three beats.

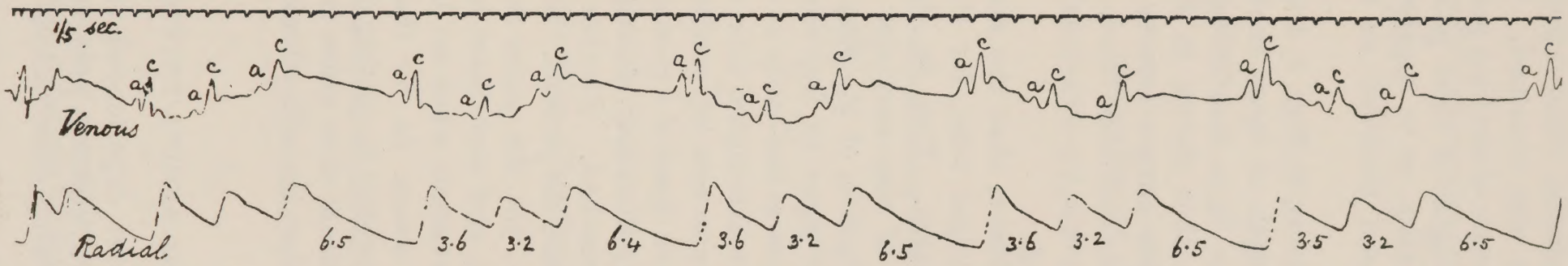

Fig. 4. × $\frac{7}{8\cdot5}$ linear. The same, showing a respiratory relationship. The pauses occur regularly after 3 beats. As in the last two figures there are definite numerical relationships between the lengths of the beats.

in the jugular pulse. Cardiograms show no trace of any beats other than those depicted, and a precisely similar observation applies to long electrocardiographic curves which were also obtained. The question which first offers itself for discussion is the nature of the long pauses, treated individually. The definite relationship of the lengths of these pauses to those of preceding beats is so frequent and so constant, as to suggest the presence of a sino-auricular block, such as has been recorded as the interpretation of an irregularity by Wenckebach[3].

In examples, such as those shown in Figs. 3, 4 and 5, such an explanation would suffice. The increased length of those beats which directly succeed the longest pauses* may be due to a shortening of the preceding Ss-As interval. In examples, such as that shown in Fig. 7, the interpretation will not hold, and curves of this nature convey a doubt as to whether we are justified in assuming sino-auricular block, even in clear instances where in an otherwise regular rhythm an occasional dropped beat is present, auricle and ventricle participating.

We cannot escape the conclusion that the mechanism in this instance is the result of alterations of vagal tone, for the irregularity is usually entirely subservient to respiration. This in itself would not exclude the possibility of a depression of sino-auricular conductivity as a basal factor in the mechanism of production of the long pauses. At times, as we have seen, the irregularity may occur in the absence of breathing; this likewise does not exclude the vagus as the cause of the varying lengths of the beats. A direct test was therefore attempted soon after the admission of the patient and at a time when the pulse rate lay consistently between 40 and 52 per minute; belladonna was given in doses of 5 minims of the tincture at intervals of 4 hours, and was steadily increased during the next few days until a dosage of half a drachm was reached. Further than this the dose could not be carried, as the drug distressed the patient. But, though the long pauses were never absent at any time, yet the pulse rate increased at frequent intervals to rates of 80 and 100, and such rates had never been met with during the first five days of observation, when no drugs were employed.

The conclusion, therefore, seems to be that the doses of atropine administered were distinctly influential in cutting down the frequency of the long pauses responsible for the usual slow pulse rate; and the observation supplies confirmatory evidence that the arrhythmia as a whole was of vagal origin.

Attacks of angina pectoris.

While in hospital the patient experienced several attacks of angina pectoris, and the account of her symptoms previously given was confirmed. Seen in several of them, the following observations were made:—The earliest

* A similar phenomenon is well seen in a parallel and experimental irregularity recorded by Hering (*Zeitschr. f. exper. Pathol. u. Therap.*, 1906, III, *Fig.* 3*K*).

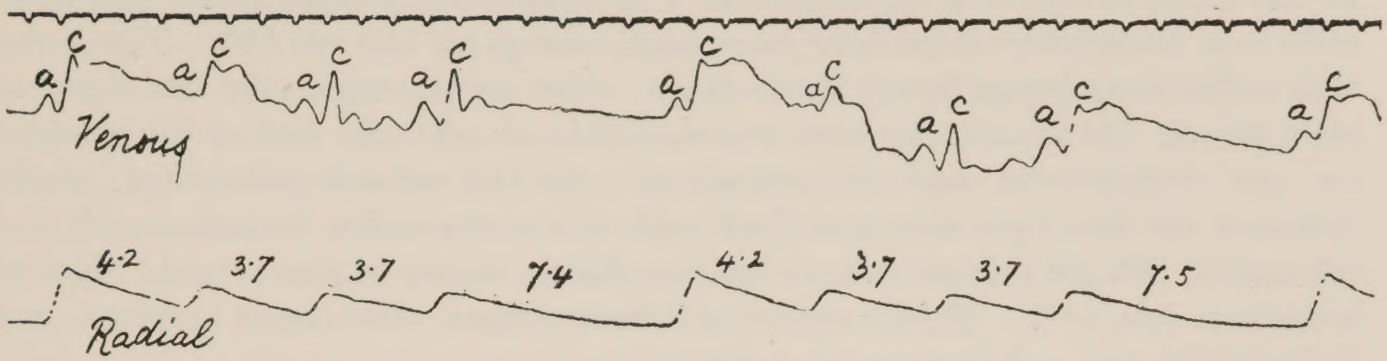

Fig. 5. × $\frac{5}{5 \cdot 5}$ linear. The same; the long pauses occur at each fourth beat. Numerical relationships are very exact.

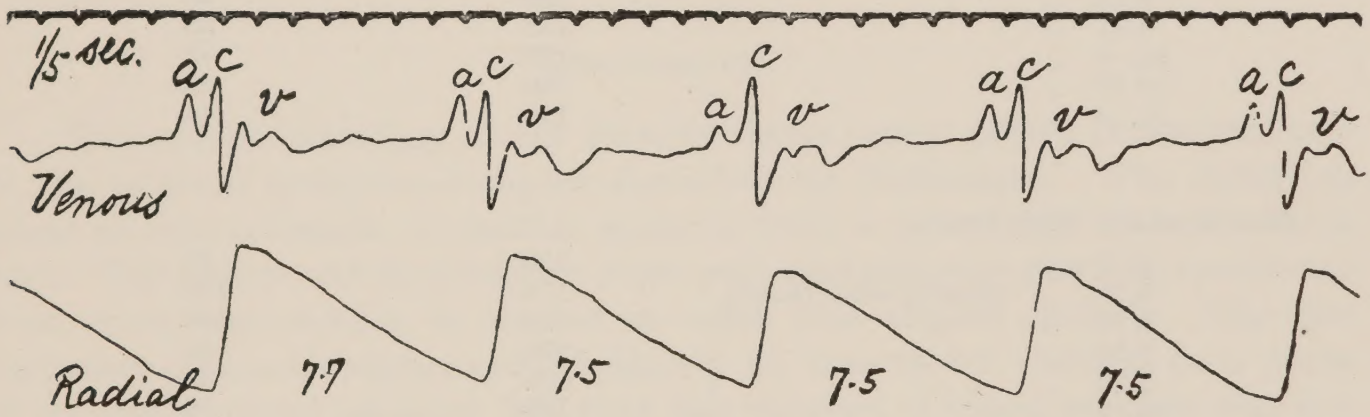

Fig. 6. A curve consisting of long beats only; suspended respiration.

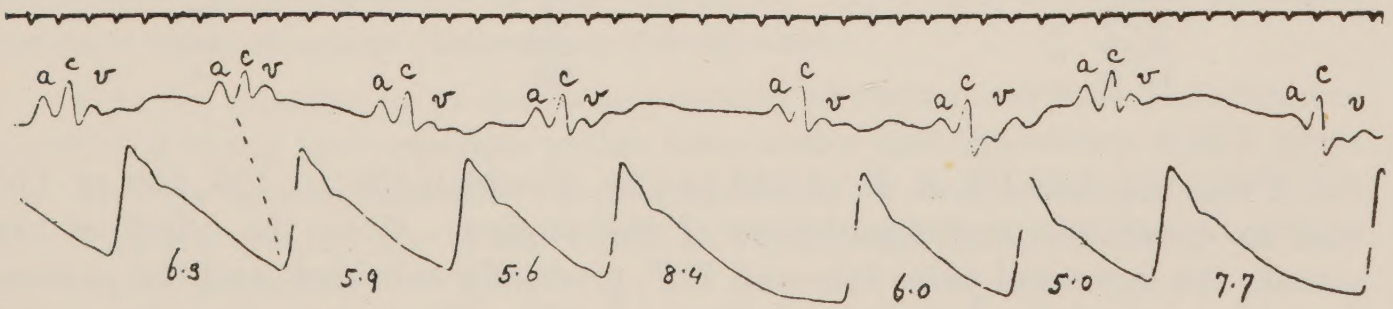

Fig. 7. × $\frac{5}{6 \cdot 3}$ linear. An example of an arrhythmia occasionally shown. The lengths of pauses show no definite numerical relationship.

sign of onset was the blanching of the face and extremities, and it was accompanied by great precordial distress and urgent dyspnœa. With the increasing distress the patient often became speechless and rigid, clutching to the sides of the bed for support. The expression was anxious, the pulse rate was invariably markedly increased, rising to 110 or 146. The pulse was sometimes irregular at these rates ; but no tracings were obtained at such times. As a rule, the rate was maintained until the end of the attack ; on one occasion it was not sustained. As the attack proceeded, slight cyanosis of the lips was manifest and the extremities became cold and clammy. Blood pressure observations during many of the attacks showed constant results*. Three series of observations, the most perfect, are included in the accompanying table :—

First attack (6th June).

Time.		Blood-pressure.	Pulse.
7. 0 p.m.	..	114 ..	44
9.20 ,, onset	..	114 ..	10
9.23 ,,	..	154 ..	68
9.26 ,,	..	150 ..	68
9.31 ,,	..	150 ..	68
9.37 ,,	..	142 ..	50
10. 0 ,,	..	140 ..	52
10.15 ,,	..	130 ..	40
11. 0 ,,	..	130 ..	44

Second attack (12th June).

7. 0 p.m.	..	130 ..	56
9.10 ,, (shortly after onset)		170 ..	132
9.15 ,,	..	170 ..	140
9.20 ,,	..	130 ..	60
9.35 ,,	..	120 ..	52
10. 0 ,,	..	120 ..	56

Third attack (14th July).

7. 0 p.m.	..	130 ..	46
10.22 ,, (onset)	..	130 ..	124
10.25 ,,	..	164 ..	146
10.28 ,,	..	168 ..	140
10.31 ,,	..	168 ..	72
10.34 ,,	..	124 ..	50
10.45 ,,	..	124 ..	54
11. 0 ,,	..	120 ..	54

From the usual S. B. P. of 110 to 130, a sudden rise to 150, 160 or 170 was an invariable accompaniment of the angina. With the offset of the attack, the increased pulse rate and B.P. gradually subsided, and the patient lay in a semi-collapsed state, shivering violently. The temperature was never raised. Subsequent to the attack, exhaustion and headache were frequent.

* The rise of blood-pressure was not the result of the rigidity before mentioned. The limbs were relaxed at the times when the observations were made.

Cutaneous tenderness was almost universal, but was specially marked over the left chest, in the neck, down the inner surface of the left upper arm and the radial border of the same forearm. Deep tenderness was also noted in the pectorals and deltoid of the left side. Pressure in the intercostal spaces in the left axilla could not be borne, and frequently the sterno-mastoids were too sensitive to allow the application of the polygraph receiver. Otherwise nothing abnormal was noted. The heart was never dilated during the attack, the percussion limits being identical with those preceding it.

The patient has more recently submitted to operation, in which a partial hysterectomy was performed. Subsequent to the operation, the pulse was frequent and regular for long intervals and for several days. As a rule, in long strips of curve, only occasional pauses were recorded. The length of a pause was always exactly double that separating two normal beats. During the whole of this time the patient complained in a minor degree of precordial anxiety and of tenderness of the skin areas. On one or two occasions definite anginal attacks occurred. Later, these subsided and the original pulse irregularity returned.

Discussion.

The symptomatology of the attacks shows many points of resemblance to the original symptom-complex described by Nothnagel.[2] The radiating pains so characteristic of cardiac seizures form a prominent feature of this case. The nature and distribution of the pain and its accompanying symptoms is in every way similar to that of so-called true angina pectoris. The disturbances in the extremities point definitely to vaso-motor changes as a cause of the rise of blood pressure, but that this increase of blood pressure was not necessarily the cause of the attacks is shown by the fact that the patient was well aware of her danger before the rise of pressure took place. It is possible, therefore, that in this instance it was rather the result than the cause of the seizure, or that pain and augmented pressure arose from a common cause. The increase of pulse rate during the attacks may be attributed to an abeyance of that increased vagal tone to which has been assigned the customary diminution of heart rate.

It is the possible or probable association of central nervous disturbances conducted along two separate paths, vaso-motor and inhibitory, which gives this case its special interest. It is impossible to assert definitely in what way they are related one to the other, or to the underlying cardiac condition previously described.

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[2] Nothnagel. Deutsch. Archiv f. klin. Med., 1867, III, 309.

[3] Wenckebach. Archiv f. Anat. u. Physiol., 1906, Phys. Abth., 297.